REMOVAL OF THE LARYNGEAL MASK AIRWAY

IN CHILDREN:

DEEPLY ANAESTHETIZED COMPARED

WITH AWAKE

Dissertation submitted in partial fulfillment of the

requirements for the degree of

M.D. (Anaesthesiology)

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CERTIFICATE

This is to certify that **Dr.D.ASHOK KUMAR** , has prepared this dissertation titled **"REMOVAL OF THE LARYNGEAL MASK AIRWAY IN CHILDREN : DEEPLY ANAESTHETIZED COMPARED WITH AWAKE"** under my overall supervision and guidance in Madras Medical College, Chennai in Partial fulfillment of the regulations of The Tamilnadu Dr.M.G.R. Medical University, for the award of M.D. degree in Anaesthesiology.

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CONTENTS

INTRODUCTION

Airway management is one of the most important skill in the field of anaesthesiology, Inability to secure the airway can lead to catastrophic events. Before 1990 the face mask and the endotracheal tube were the only available airway devices. Since then several supraglottic airway devices have been developed of which the LMA is the most popular one. The laryngeal mask air way is a supraglottic device that is designed to provide a seal around the larygneal inlet.

As proved in many studies extubation is associated with changes in cardiovascular responses and adverse respiratory events. Laryngeal mask airway insertion and removal also produces adverse respiratory events such as coughing and straining oxygen desaturation, bronchospasm, Iaryngospasm and excessive salivation in less severity

Daley et al documented that in hyperreactive airway, deep tracheal extubation was preferred by 64% anaesthesiologist.

Valley rd etal and Pander Dr etal proved through Independent studies extubation of endotracheal tube can be safely performed in peadiatric patients maintained with volatile anaesthetics. Eugene et al. also prove deep tracheal extubation is safe.

Several studies showed that LMA removal is also most similar to tracheal extubation. Baird MD et al found that LMA removal in deeply anaesthetized state is associated with less Oxygen desaturation events , than the LMA removal in awake state. Gataure et al showed that it is safe to remove LMA in deeply anaesthetized state. These studies went on to prove that LMA removal in deeply anaesthetized state is less likely to be associated with adverse respiratory events while maintaining adequate airway patency and Oxygen saturation.

Patel et al concluded that choice of extubation technique may be dictated by the surgical procedure. Kitching et al studied adverse respiratory events occurring in awake or deep removal of LMA and found that coughing and straining to be the major respiratory event occurring when awake removal is done. So, this study was done to evaluate the advantages of deeply anaesthetized removal of LMA in some specific surgeries such as Uretheroplasty.

AIM OF STUDY

The purpose of this study is to compare the incidence of undesirable respiratory events when the LMA is removed from pediatric patients who are fully awake or from patients who are deeply anaesthetized.

The adverse respiratory events that are compared

1. Coughing and straining
2. Oxygen de-saturation
3. Laryngospasm
4. Bronchospasm
5. Vomiting and excessive salivation

BASIC SCIENCES

ANATOMY OF PEDIATRIC AIRWAY

In infants and young children the head is relatively large and the neck is shorter than in the adult. These factors together with the relatively large tongue predispose to upper airway obstruction and probably accounts for the greater use of tracheal Intubations in these patients.

The Infant glottis is situated opposite to C_3-C_4 intervertebral disc. By the age 3 years it has descended to the C_4-C_5 Interspace, where it remains until puberty, when it descends again to lie opposite the body of C_5. The epiglottis of the infant is longer and U-shaped posterior as opposed to the flat leaf shape of the adult.

Infant larynx occupies a more anterior position compared with that in adult.

The larynx is funnel shaped in children below 8 years of age with the narrowest portion being at the level of the cricoid cartilage. The vocal cords of the neonate are slanted such that the anterior commissure, is more caudal than the posterior commissure.

Applied Anatomy

Anatomical structures relevant to laryngeal mask airway space include the mouth, oropharynx, laryngopharynx and hypopharynx.

Mouth

The roof of the mouth formed by vaulted palate comprising the bony hard palate (anterior 2/3) and soft palate (posterior 1/3). The general shape of hard palate is such that a food bolus is directed into the oropharynx inlet with the stiffened soft palate shielding the nasopharynx. There may be some difficult in passing the laryngeal mask airway into oropharynx, particularly if the angle of approach between the hard palate and posterior oropharyngeal wall is less than 90 degree. Mouth opening is essential for laryngeal mask airway placement. The average distance between the upper and the lower incisor teeth in adult patients with normal temporo mandibular joint function is 47 mm with a range of 31-55 mm. It is possible to insert the laryngeal mask airway with an incisal opening of 12 mm.

Oropharynx:

To enter the laryngopharynx the laryngeal mask airway passes through the oropharynx. This is bounded is anteriorly by the palatoglossal arch and the tongue. Immediately posterior to the tongue is the epiglottis, a leaf shaped plate of elastic cartilage covered with mucous membrane. The median glosso epiglotic fold runs between the front of the epiglottis and the back of the tongue and on each side is a lateral depression, the epiglottic valleculae. The palatine tonsils lie in the lateral walls of the oropharynx and if grossly enlarged may impede passage of the laryngeal mask airway. The anterior aspect of the cervical vertebrae covered in muscle and mucosa bound the posterior part of the oropharynx.

Laryngopharynx and hypopharynx

The posterior and lateral walls of laryngopharynx are bounded by the inferior and middle constrictor muscles, which tapers rapidly towards the esophagus. The anterior wall is formed by the inlet of the larynx superiorly and the pyriform fossae, the interarytenoids muscles, the posterior cricopharyngeus muscle and the attachment of the longitudinal muscles of the esophagus. The hypopharynx is the space behind the arytenoids and cricoid cartilages. It is approximately 3.5 cm in length. The superior 1.5 cm lies behind the arytenoids cartilage's and the inferior 2 cm lies behind the cricoid cartilage.

Neurovascular considerations:

Several nerves and blood vessels within the tissues of the oropharynx may theoretically be compressed by mal-position and or over inflated laryngeal mask. Examples are the lingual artery as it enters the base of the tongue, the glossopharyngeal nerve as it passes between the superior and middle constrictor muscles; the recurrent laryngeal nerve as it enters the larynx by passing deep to the lower border of the inferior constrictors; and the lingual nerve as it enters the mouth below the inferior border of the superior constrictor and continues against the periosteum of the mandible posterior to the third molar.

PHYSIOLOGY

Physiological implications:

Gastrointestinal System :

The Swallowing reflex:

Laryngeal mask airway insertion is highly successful as a blind technique as it utitilizes the normal existing physiological mechanism of swallowing to follow the natural curve and the direction of the upper airway; the inserting finger imitating the tongue action in swallowing food. However, swallowing reflex itself must be suppressed for the insertion and the tolerance of the laryngeal mask airway cuff in the pharynx. Insertion of the laryngeal mask airway in the non-anaesthetized patient triggers a variety of protective and digestive reflexes including coughing, gagging, retching, swallowing and hypersalivation. With increasing anesthetic depth these reflexes are suppressed to a varying degree. If laryngeal mask airway insertion occur at inadequate anaesthetic depth a single incomplete swallow may occur and the glottis, instead of transiently closing as in the normal swallow remains closed for a period of 20-30 sec, leading to a misdiagnosis of laryngeal spasm or malposition. Coughing is more likely to occur if the laryngeal mask airway tip impacts with the glottic inlet, but may also occur if pharyngeal secretions are driven into the glottis by the mask as it is

inserted. The ability to utilize the swallowing pathway for insertion, which does not normally require a person to assume a different head position, makes possible the successful use of the laryngeal mask airway in patient in whom movement of the cervical spine is impossible or contra indicated due to pathology, stiffening the bony architecture or endangering the spinal cord.

The esophagus:

The presence of mask in the pharynx and stimulation provoked by insertion inevitably involve the upper gastrointestinal tract reflexes. The sustained involve the upper gastrointestinal tract reflexes. The sustained distension of the pharynx induces prolonged relaxation of the lower esophageal sphincter.

The Pharynx contains mechano- and chemoreceptors, which play part in triggering the primary peristaltic wave of deglutition. However, inappropriate stimulation or by passing of these trigger zones may produce a less coordinated response including secondary peristalsis which lacks both the speed of the completion and the co-ordination of primary peristalsis and can result in relaxation of the lower esophageal sphincter without subsequent immediate restoration of tone. A confounding factor in the interpretation of pH studies comparing the laryngeal mask airway and face mask is that the presence of the mask blocks passage of alkaline secretions from the upper pharynx reaching the lower esophagu. One factor preventing aspiration may be persistent function of the upper esophageal sphincter Vanner et al in 1992 (showed that during spontaneous ventilation anaesthesia, upper esophageal sphincter pressure does not fall significantly with an laryngeal mask airway

in situ, suggesting that the pharyngo-upper oesophaeal sphincter contractile reflex may be intact further upper oesophageal sphincter is known to constrict in response to the presence of acidic fluids in the lower oesophagus.

Pharyngeal mucosa:

The inflated laryngeal mask airway cuff could generate sufficient compression and shearing forces to cause a reduction in pharyngeal mucosal blood flow and direct tissue trauma, as can occur during endotracheal intubation. Some parts of the pharynx compressed against rigid tissues such as the hyoid bone or cervical vertebrae may be more susceptible to damages. The pharynx however, is a highly distensible structure, which is normally subjected to large transient pressure changes and distortion under many physiological conditions. Marjot in 1993 has shown that calculated transmitted mucosal pressures potentially exceeds the capillary perfusion pressure of the adjacent pharyngeal mucosa, but there is no evidence that this is harmful even over prolonged periods. No major pharyngeal trauma has been reported in several million laryngeal mask airway anasethetic and minor morbidity, such as sore throat, is usually mild and is less than for the endotracheal tube and similar to the face mask. To avoid pharyngeal mucosal damage it has been postulated that either the pressure on the pharyngeal mucosa must be lower than calculated values or the pharyngeal mucosa must be resistant to ischemic damage or adaptation of the pharyngeal blood vessels must occur either due to uneven distribution of pressures exerted by the laryngeal mask aiway or to a redistribution of blood

flow. A futther possible consequence of pharyngeal mucosal trauma is transient bacteremia, but, in contrast to endotracheal intubation, this does not appear to occur during laryngeal mask airway insertion.

Respiratory System

The laryngeal mask airway should cause minimal triggering of or interference with the lung defences because they are distal to the device. Support for this idea comes from clinical data which suggests that laryngeal spasm, bronchospasm, and coughing occur less frequently than with the endotracheal intubation. Endotracheal intubation is known to interfere with mucociliary clearance and the laryngeal mask airway does not impede mechanically, although anaesthesia itself may interfere with ciliary function. It is thus possible that the laryngeal mask airway may be of benefit in maintaining the tracheobronchial climate and this may have implications for patients with pulmonary pathology. There is clinical evidence that it may offer benefit in patients with respiratory diseases. For instance it has been suggested that paediatric patients with mild upper respiratory trace infections may have improved postoperative oxygen saturation with laryngeal mask airway compared with the endotracheal tube

The laryngeal mask airway by passes the narrow laryngopharyngeal space providing an unobstructed low resistance airway Dead space is approximately 50% less than with the facemask but more than when the trachea is intubated.

Reigneer et al in 1995 have shown that in children aged 6-24 months anaesthetized with halothane, there is less paradoxic inspiratory movement breathing through the larynegeal mask airway than endotracheal tube. It has also been shown that laryngeal mask airway can accommodate a larger fiber optic scope than either the flexible laryngeal mask airway or endotracheal tube while permitting an acceptable ventilatory flow.

Cardiovascular system

Wilson I.G. et al in 1992 and Watcha MF et al in 1991 have shown that insertion of the laryngeal mask airway is associated with only a 0-20% rise in blood pressure and heart rate in both adults and children. Laryngoscopy and subsequent endotracheal intubation are associated with a 25-50% rise in blood pressure and a similar increase in heart rate. Plasma concentration of adrenaline and nor adrenaline are higher following endotracheal intubation than laryngeal mask airway insertion. Patients with laryngeal mask airway require significantly less anaesthetic agent to maintain depth of anaesthesia.

LARYNGEAL MASK AIRWAY

The LMA was conceived and designed by dr. Archie Brain in U.K. in 1981. Following prolonged research, it was released in 1988. At an early stage in its development, the inventor realized its potential in the management of the difficult airway.

Today, it has a clearly established role as an airway device in the elective setting where neither the procedure nor the patient requires tracheal intubation. It has now become an established part of routine airway management and has proved extremely useful in managing the difficult airway**.**

Concept and design

The LMA fills a niche between the face mask (FM) and tracheal tube (TT) in terms of both anatomical position and degree of invasiveness. It is manufactured from medical grade silicone rubber and is reusable.

It consists of 3 main components. An airway tube, inflatable mask and mask inflation line. The airway tube is slightly curved to match the oropharyngeal anatomy, semirigid to facilitate atraumatic insertion and semitransparent, so that condensation and regurgitated material is visible. A black line runs longitudinally along its posterior curvature to aid in orientation. The distal aperture of the airway tube opens into the lumen of an inflatable mask and is protected by two flexible vertical rubber bars, called mask aperture bars (MAB), to prevent the epiglottis from entering and obstructing the airway.

The inflatable mask is oval shaped with a broad, round proximal end and a narrower, more pointed distal end. It has an inflatable cuff and a semirigid, concave, shield like backplate. The cuff is attached to the outer rim of the backplate.

The inner aspect of the mask is called the bowl, which is comprised of the distal aperture, mask aperture bars, backplate and the inner aspect of the inflatable cuff.

The mask inflation line, which is attached to the most proximal portion of the cuff in the midline consists of four parts, the long narrow inflation line itself, the inflation indicator balloon (pilot balloon), a metallic valve and the syringe port. The valve, which has a white coloured core is made from polypropylene and has a stainless steel spring valve. The LMA is available in eight sizes, from neonates to large adults, 1 to 6 and two half sizes 1.5 and 2.5. The cuff, but not the tube, has identical proportions among sizes; it gets about 155 larger for each size.

Table - 1 : Classic LMA Specifications		
Mask size	Patient weight (kg)	Maximum inflation volume (mg)
1	< 5	4
1.5	5 - 10	7
2	10 - 20	10
2.5	20 - 30	14
3	30 - 50	20
4	50 - 70	30
5	70 - 100	40
6	>100	

Anatomy

The cuff is pressed against several structures in sequence - the hard palate, the soft palate, the naso/oropharyngeal and then the hypopharyngeal portion of the posterior pharyngeal wall.

The ideal final anatomic position occupied by the classic LMA is as follows:

The distal most cuff sits in the hypopharynx at the junction of the upper oesophagus and respiratory tracts, where it forms a circumferential

low pressure seal around the glottis. Superiorly, the upper part of the mask lies under the base of the tongue, allowing the epiglottis to rest within the bowl of the mask at an angle probably determined by the extent to which passage of the mask has deflected it down - wards. When inflated, it lies with the tip resting against the upper esophageal sphincter, the sides facing the pyriform fossae with the upper surface behind the base of the tongue and the epiglottis pointing upwards. The aperture of a properly positioned LMA aligns itself anatomically with the laryngeal inlet.

The tip of the LMA cuff lies at a variable depth behind the cricoid cartilage; and the posterior surface immediately anterior to the C_2 to C_7 cervical vertebrae. The laryngeal inlet can be tipped anteriorly by the inflated LMA cuff when cricoid pressure is applied; this may explain why blind intubation via the LMA is more difficult with cricoid pressure applied.

Indications

- Elective short surgical procedures under general anaesthesia excluding head and neck surgery.

- Rescue airway in "cannot intubate - can ventilate" and "cannot intubate, cannot ventilate" scenario if the problem is supraglottic in nature, since successful use of the LMA does not require the constellation of factors required for direct laryngoscopy and tracheal intubation. In 1998 it entered the American Society of Aneshesiologists' difficult airway algorithm in five different places,

both as a ventilatory device (airway) and a conduit for endotracheal intubation.

- Cardiopulmonary resuscitation.

Contraindications

- Mouth opening less than 1.5 cm
- Poor lung compliance
- Airway pressure more than 20cm of H_2O
- Non fasting patients

Insertion technique

LMA insertion can be considered in context of swallowing both in terms of the space it occupies and the type of reflex response it elicits. The insertion technique does not require the use of a laryngoscope or muscle relaxants and is designed to imitate the mechanism whereby the food bolus is swallowed.

Preparation of the LMA and the patient is essential for successful placement. A selection of LMA sizes should be available in addition to the one most likely to fit because the anatomical features of the larynx cannot always be predicted from the physical examination. Most of the induction agents can be used to facilitate placement of the LMA. Insertion LMA can

be done with volatile anaesthetics also. The adequate depth of anaesthesia for LMA placement is significantly less than that for tracheal intubation.

Several insertion techniques have emerged to complement the original technique which was described when the LMA was introduced. The standard technique involves a completely deflated LMA, held like a pen guided into the pharynx with the index finger of the operator at the junction of the tube and the bowl, with the operator at the head of the patient and the LMA aperture facing caudaly, With the head extended and the neck flexed by using the hand under the occiput, under direct vision, the tip of the cuff is pressed upwards against the hard palate. The LMA is advanced into the hypopharynx till a resistance is felt. The cuff is then inflated with just enough air to seal, to intra cuff pressure around 60 cms H_2O. A common alternative technique popular in children described by McNicol, consists of inserting a partiality inflated LMA into the pharynx above the epiglottis with the aperture facing cranially, the LMA is then turned 180 degrees before advancing it into its final position.

The LMA should then be secured after insertion in such a way, so as to prevent rotation and movement cranially. If surgical access allows, a preferred way to connect the LMA to the anaesthesia circuit is to direct the circuit connection caudally and bring the circuit limbs down on the side of the patient's neck and head.

Signs of correct LMA placement

a. Slight outward movement of the tube upon LMA inflation.

b. Presence of a small oval swelling in the neck around the thyroid and cricoid area.

c. No cuff visible in the oral cavity.

d. Expansion of chest wall on bag compression.

Before taping the LMA in place, a bite block is inserted to stablise the LMA and prevent tube occlusion.

Emergence technique

Removal of the LMA can be accomplished either during deep anaesthesia or awake state.

LMA and aspiration

Although the correctly placed LMA tip lies against the upper esophageal sphincter, the LMA does not isolate the respiratory tract from the gastrointestinal tract and does not protect the lungs from regurgitated gastric contents. The glottic seal is usually lost at peak airway pressures above 20 cms H_2O. Incidence of aspiration with the LMA is 2 per 10,000.

Fentanyl

It is a synthetic phenylpiperdine - derivative opiod agonist that is structurally related to meperidine.

Dose of 1-2 :g/kg is given IV to provide analgesia. It has got rapid onset and shorter duration of action.

Atropine

It is used as premedication to protect the heart from vagal reflexes and to prevent excessive salivation.

During anaesthesia that includes a volatile drug the does of atropine needed to increase the heart rate may be decreased perhaps reflecting depression of vagal centres during anaesthesia.

Halothane, like opiods may increase the central vagal tone aceounting for the greater heart response after administration of atropine to patient anaesthetized with halothane.

HALOTHANE

History

The development of halothane was facilitated by advances in fluorine chemistry made in the course of the development of unclear weapons during the Second World War, although it was not itself synthesized until 1951, by Suckling of ICI, with the first clinical trails reported in 1956.

Physical chemistry

Molecular weight	197.38
Boiling point (^{O}C)	50.2
Vapour pressure at 20 ^{O}C (kPa)	32.53
Maximum vapour concentration at 20 ^{O}C	32%

At atmospheric pressure halothane is a colourless clear volatile liquid with a non-irritant vapour.

Chemistry

Although non-inflammable in air at any concentration, 4.75% halothane vapour will ignite in a 30% oxygen-70% nitrous oxide mixture. In pure nitrous oxide, 1% halothane will ignite. In practice, however, halothane may be regarded as non-inflammable in clinically used gas mixtures.

The halothane molecule, an alkane, is less stable than enflurane or isoflurane, and requires the addition of 0.01% thymol as a stabilizer. it must also be kept cool and protected from UV light. Although there is theoretical possibility of accumulation of breakdown products of halothane in a closed

circuit, they are only present in very low concentrations and are not though to represent a risk. In the presence of moisture, halothane vapour is mildly corrosive to aluminium, tin and brass.

Anaesthetic characteristics

Blood:gas partition coefficient	2.3 ($37\,^{O}C$)
Oil: gas partition coefficient	224 ($37\,^{O}C$)
MAC (%)	0.75

Halothane has a non-irritant vapour allowing the administration of high concentrations with minimal risk of coughing, breath-holding or laryngospasm. In terms of speed and smoothness of induction of anaesthesia this outweighs the lower blood-gas solubility coefficients of enflurane and isoflurane, particularly in inhalational induction of anaesthesia in children.

Halothane possesses no significant analgesic properties.

Circulation

Halothane is a profound myocardial depressant, producing a dose-related fall in arterial pressure. Cardiac output, stroke volume, left ventricular stroke work and myocardial contractility are all depressed and right atrial and left ventricular end-diastolic pressure are increased. Heart rate may decrease but systemic vascular resistance is unchanged. *In vitro*

studies confirm that the major site of action of halothane on the circulation is the myocardium itself, the mechanism being a reduction in intracellular Ca2+ by inhibition of ion transport in the myocardial sarcoplasmic reticulum, possibly with a reduction in responsiveness of the myofibrils to calcium. Meanwhile, there is a depression of baroreflex control of systemic vascular resistance and of heart rate, allowing vagal predominance and bradycardia (which is easily reversed by atropine).

In Halothane anaesthesia, junctional rhythms is relatively common. Halothane slows conductive tissues allowing escape and re-entry type phenomena, as well as increasing automaticity of the myocardium and sensitizing it to the arrhythmogenic effects of catecholamines, endogenous or exogenous. Premature ventricular exrasytoles are often seen, and high circulating levels of catecholamines can occasionally precipitate ventricular tachycardia or fibrillation.

Although the overall systemic vascular resistance is unaltered by halothane, there are marked changes in the distribution of cardiac output. Flow to the skin and the cerebral circulation is increased, splanchnic, renal and hepatic flow decreased, particularly during hypotension. The coronary circulation is not directly affected.

Respiration

Spontaneous respiration under halothane anaesthesia is rapid and shallow. In common with enflurane and isoflurane, halothane depresses the

ventilatory response to both hypercarbia and hypoxia, effects that seems to be mediated largely on the respiratory centres themselves, although halothane does exert an action on the carotid bodies. In addition to its effects on the neural control of respiration, halothane inhibits hypoxic pulmonary vascoconstriction.

Halothane is a powerful bronchodilator, and is a drug of last resort in the treatment of status asthmaticus. Its antagonism of histamine-induced bronchospasm may persist for 24 hr following anaesthesia. Although ciliary movement is depressed by halothane, recovery of function is rapid.

Central nervous system

Halothane has long been known to increase cerebral blood flow and thus intracranial pressure, particularly in the presence of low intracranial compliance. At normocarbia, 1 MAC of halothane will increase cerebral blood flow by 150%; 2 MAC will quadruple it. Hyperventilation greatly reduces this effect, but hypocarbia must be established before the introduction of halothane. Autoregulation of the cerebral circulation is abolished by 1 MAC halothane.

Other systems

In common with enflurane and isoflurane, halothane is a significant skeletal muscle relaxant, and will potentiate the effects of neuromuscular blocking agents, apparently by a postsynaptic action. In patients with myasthenia gravis, depression of the train-of-four response may be seen.

Halothane is a uterine relaxant and may contribute to uterine laxity and increased blood loss after delivery at Caesarean section or following therapeutic abortion. In the past, worries about haemorrhage and depression of the fetus led to the use of very low concentrations of halothane in obstetric patients, with a resulting high incidence of awareness. It is now appreciated that at inspired concentrations of up to 0.5% halothane does not materially affect blood loss, and fetal depression blamed on anaesthetic agents may have been due to undiagnosed aortocaval compression.

Higher concentration of halothane may be a useful uterine depressant in anaesthesia for external version of the fetus, and have been used to relax the uterine constriction ring, although there is a considerable risk of hemorrhages, and the concentrations needed may produce undesirable hypotension.

It is well known that MAC, the minimum alveolar concentration required to prevent movement in response to surgical incision in 50% of patients, decreases with age.

Regression analysis showed that data for humans derived from a comprehensive literature survey were consistent, for age > 1 yr, with log MAC decreasing with increasing age at the same rate for all inhaled anaesthetics; approximately equivalent to 6% change per decade of age.

With some slight reservation on difference between data from different institutions, the present data for humans are consistent (for age > 1 yr) with the equation $MAC = a \times \text{Log } 10^{bx}$ where x = difference in age (in

years) from 40, b = 0.00269 (95% confidence limits (CL) -0.0030, -0.0024) and a = MAC at age 40 yr, which, for anaesthetics currently in use clinically, is given by halothane 0.75%, isoflurane 1.17%; enflurane 1.63%; sevoflurane 1.80%; desflurane 6.6%; nitrous oxide 104%; with 95% CL of approximately ±7% (±10% for desflurane, ±17% for enflurane).

Halothane adjusted for Age

Mean Age (Years)	MAC %
0.2	**1.08**
1.5	**0.97**
4.1 **8.4**	**0.91** **0.8**

Metabolic fate

In vivo, at least 20% and possibly as much as 40% of absorbed halothane undergoes biotransformation to a variety of metabolites, including trifluoroacetic acid. This process is enhanced by enzyme induction, and is thought to hold the key to halothane hepatotoxicity.

Because halothane sensitizes the myocardium to arrhythmias secondary to exogenous catecholamines, the total dose of exogenous

epinephrine used for vasoconstriction during halothane anaesthesia is usually limited. The does necessary to cause arrhythmias has been demonstrated to be 1.4 to 2.0 :g/kg SC in adults. Children tolerate higher doses of epinephrine during halothane anaesthesia than do adults (6 to 10:g/kg); tachycardia and hypertension are more common side effects than arrhythmias. It is reasonable to limit the total dose of epinephrine added to local anesthetic or saline to 10 :g/kg/20 min.

Hepatic Toxicity

Although halothane-associated toxicity has been reported, there is little evidence of a clinically important incidence of hepatotoxicity secondary to halothane in the pediatric age group. Several retrospective pediatric studies suggest that the incidence of halothane - associated hepatitis ranges from 1 in 82,700 to 1 in 200, 155 anesthetic procedures. A prospective examination of 1362 halothane anesthetic procedures in 186 children found minor increases of hepatic enzymes, no cases of jaundice, and several cases of elevated enzyme levels postoperatively in patients with preoperative enzyme abnormalities. Papers reviewing halothane-associated hepatitis have reported a low to nonexistent incidence of "unexplained" hepatitis following exposure to halothane in pediatric patients. Despite the very low incidence of halothane-associated hepatitis, the serum of some of these patients was positive for antibodies to rat halothane-sensitized hepatocytes. Out of millions of halothane anesthetics of children, only several pediatric deaths have been reported.

Another report summairzed the British experience of seven pediatric patients specifically referred with the possible diagnosis of "halothane hepatitis"; one patient died, and the other six recovered uneventfully. The serum from these patients was collected during a 7-year period. Three different tests for halothane antibodies were used to make the presumptive diagnosis. The investigators concluded that repeat exposure to halothane should be avoided in children.

Although there is evidence for the existence of "halothane hepatitis" in adults and although animal models have been developed with several postulated mechanisms (hypoxia, toxic metabolites), the true incidence of halothane-associated hepatitis in the pediatric population remains unclear. Because children have fully developed immunologic mechanisms are capable of metabolizing halothane.

The British report's recommendation to avoid repeat halothane administration to children was made without the input from senior pediatric anesthesilogists and is inconsistent with the clinical experience of the majority of the world's major pediatric centers. In fact, there is a higher incidence of fatal hepatic dysfunction in children who have received acetaminophen than in those exposed to halothane.

There are no data to suggest that patients with pre-existing liver disease have an increased propensity to develop halothane-induced hepatic dysfunction. The true incidence of halothane-associated hepatitis in children is unknown; if halothane-associated hepatitis exists in the pediatric age group, the incidence is exceedingly low and significantly lower than it is in

adults. Because only two or three pediatric patients have been reported to have had fatal "halothane hepatitis" despite many millions of pediatric administrations, the proven efficacy, vast experience, safety, superiority for suppressing airway reflexes for deep intubation with spontaneous respirations in patients with difficult airway anatomy, and ease of acceptance by a patient with difficult airway anatomy, and ease of acceptance by a patient population that often requires a smooth mask induction to anaesthesia, halothane may still be the inhalation agent of choice for many children. The pungent smell of isoflurane, enflurane, and desflurane makes these agents less acceptable to awake pediatric paitents, especially those with a compromised airway.

Uptake and Distribution

The uptake and distribution of inhalation agents are more rapid in infants and children than in adults. This effect may in part be related to their more rapid respiratory rate, increased cardiac index, and the distribution of a larger proportion of the cardiac output to vessel-rich or well-perfused organs. These factors results in a more rapid increase in the partial pressure of inhalation agents in mixed venous blood; the more rapid uptake is one of the factors contributing to the ease of producing myocardial depression in infants and children. This more rapid uptake may also account in part for the higher incidence of cardiac arrests in children compared with adults.

Ventilation/perfusion mismatch, airway obstruction, and perhaps intracardiac defects (primarily right-to-left shunts) result in a slower rate of uptake and rise in alveolar concentration of the potent inhalation (soluble)

agents. More time is required to achieve an adequate plane of anaesthesia for safe laryngoscopy and intubation in children with any of the previously mentioned problems. This concept is especially important when inducing anaesthesia in a child with airway obstruction, when safe, smooth control of the airway is vital. Nitrous oxide and sevoflurane are insoluble, compared with halothane. Therefore, the effects of respiratory rate, increased cardiac index, and distribution of cardiac output should be of less clinical importance.

The rate of induction and awakening may also be related in part to the type of anesthetic circuit used. A non-rebreathing system produces a more rapid rise in alveolar anesthetic concentration than a similar concentration delivered by a rebreathing (circle) system. With a circle system, it is necessary to consider the volume of the anesthetic tubing, the carbon dioxide absorber, and the humidifier in relationship to the patient's lung volume. With a circle system (volume of 3500 mL), a change in inspired concentration takes longer to equilibrate than a similar change made with a none-re breathing circuit (volume of 1200 mL). A more rapid induction and perhaps a better control of anesthetic depth may be achieved with a non-rebreathing system. This concept is important in neonatal anaesthesia because very small changes in anesthetic concentration rapidly equilibrate with the small lung volume. A non-rebreathing system also allows more rapid elimination of potent inhalation agents. These concerns may be considered when selecting an anaesthetic circuit for use in Infants and neonates. If very high fresh gas flow is used it takes the characteristics of NON breathing system.

The pharmacokinetics of inhalational anaesthetics have been extensively studied over the last 30 years.

The first development is the practical application of new techniques for studying anaesthetic distribution *in vivo*. Although direct methods of determining drug distributions, such as autoradiography, were applied to the inhaled anaesthetics some time ago, it is only more recently that non-invasive observations have been possible. A range of unclear magnetic resonance techniques have been developed that are based on the signal analysis of F nuclei. These, of course, are applicable to all fluorinated anaesthetics and enable the kinetics of cerebral distributions of those agents to be followed directly.

The second development is the renaissance of alternative concepts in the interpretation of uptake and distribution data. The kinetics of anaesthetic uptake and distribution have been interpreted in the past as being *perfusion* limited rather than *diffusion* limited. Thus the emphasis has been on the solutbilities of the different agents and the relative blood flows and capacities of the different tissues. However, there is increasing recognition that, in addition to perfusion - based distribution, there is both intertissue diffusion of the agents as well as diffusion through the skin and other tissue-gas interfaces. Moreover, the simple fixed capacity concepts of the different compartments have to be modified to allow for the metabolism of the anaesthetic. The latter aspect is well established for the intravenous anaesthetics but has been regarded as a relatively minor effect for the

inhalation agents (in the case of uptake and distribution studies rater than toxicity investigations).

The difference between the classical and current studies on the kinetics of inhalation anaesthetics is illustrated by the water analogue.

This describes the uptake and distribution of an inhaled anaesthetic from a non-rebreathing system in terms of water flowing directly to a series of cylindrical containers. The water represents the anaesthetic itself, with the depth of water in any container representing the anaesthetic parital pressure. The containers represent the various groups of organs and tissues and their different volumes represent the storage capacities of those group for a particular anaesthetic. The sizes of the pipes connecting to the containers represents the relative perfusions of the different compartments. This hydraulic analogy can be developed of uptake and distribution of different anaesthetics.

It is clear that this model considers each compartment to be self-contained with ingress of egress of the agent via the connecting pipe recent studies have included the probability of direct interconnections between the containers representing intertissue diffusion. In addition, metabolism can be represented by a variable leak in the visceral container. Modelling the effect of metabolism is complicated by the fact that the absolute rates may only be partial pressure dependent up to a certain value, and hence the 'leak' has to be variable.

The water analogue is particularly helpful in determining the cirumstances when intertissue diffusion will become important. For example, the phenomenon could not be a significant factor during the initial uptake of the agent because the partial pressure gradients between the different compartments will be relatively small. On the other hand it will be significant when, for example, the visceral compartment is almost saturated while the other two compartments have much lower partial pressures although the absolute amounts in the compartments may or may not be comparable. Finally any intertissue diffusion will become less important towards the end of the anaesthetic uptake because the partial pressure *gradients* will again be reduced.

A similar analysis of the maximum impact of metabolism indicates that its effects will be more marked during recovery from anaesthesia. Thus comparisons between induction and education of anaesthetics may reveal more than the expected mirror image pattern.

Induction vs. Education

Nearly all the factors controlling the uptake of inhaled anaesthetics also apply to recovery from anaesthesia. Thus the characteristic uptake curves relating alveolar to inspired concentrations have their counterparts in the recovery curves relating the alveolar concentration to the alveolar concentration immediately preceding the cessation of anaesthetic administration.

The major difference between induction and education of the agent is the variable equilibration of the different tissue groups at the end of anaesthetic. Whereas at the start of induction all tissues have the same partial pressure (namely zero), at the start of education each tissue has a different degree of saturation depending on the length of the prior period of anaesthesia.

Intertissue diffusion has long been recognized as a component of uptake and distribution of anaesthetic gases but, despite some excellent mathematical modeling and direct autoradiographic evidence, the phenomenon has been regarded as non-rate limiting. The new work demonstrates that the exclusive perfusion-based models are an over-simplification and have to be replaced by perfusion-diffusion models. The diffusion component of anaesthetic distribution includes percuteaneous losses. These have measured and it would appear that the absolute magnitude of the percutaneous loss is a trivial fraction of the total amount of anaesthetic taken up. However, visceral losses from the abdomen or thorax during surgery may be considerably larger and studies are in progress to determine if this prediction is correct.

The anesthetic induction period can be an extremely stressful period for the anesthesilogist, the child, and the parents, and for observers in the area. Proper evaluation and preparation of children and their families both psychologically and physiologically is extremely important, to avoid problems that could be prevented through better planning (see Chapters 3 and 4). Failure to properly evaluate the severity of underlying medical

problems or to correct metabolic abnormalities can lead to cirtical events during the induction period. Even though a child may appear "normal" before induction, unexpected problems can occur during induction, such as laryngospasm, bradycardia, or cyanosis. Anesthesiologists who care for children must always be prepared for the unexpected. Because of the ever changing psychological and social needs of children and their parents, it is vital to be very flexible in changing to alternative methods of caring for patients and to alter plans "midstream", depending on the needs and reponses of a child despite these drawback inhalation induction technique has long, been favoured by children and paediatric anaesthescologic.

Traditional Mask Inhalation Induction

The most common method of inducing anaesthesia in children is inhalation of potent anesthetic agents through a face mask. The technique used depends on a number of factors, including the child's developmental age, understanding and ability to cooperate, and previous experiences, and the interaction of these factors with the patient's underlying medical or surgical conditions. Infants up to 8 to 10 months of age, for example, generally are not fearful of strangers and will easily separate from their parents. thus this age group does not generally require a premedication andwill usually respond to the mask. Toddlers frequently require a premedication to be able to cooperate for the smooth gaseous induction of anaesthesia. Cooperative older children generally tolerate an inhalation induction, even though they may dislike the odor of the anesthetic gas. Generally when given a choice between an intravenous or intramuscularly

induction and an inhalation induction, they will choose an inhalation induction.

The traditional mask induction of anaesthesia is accomplished by initially administering either 100% oxygen or a nonhypoxic mixture of nitrous oxide and oxygen. Although modern anaesthesia machines do not allow the delivery of hypoxic mixtures, older machines are still available that will allow this to occur. Hypoxic mixtures have no place in pediatric anaesthesia; they are potentially hazardous and unnecessary. Following several breaths of oxygen or the nitrous oxide-oxygen mixture, halothane is added, beginning at 0.25%. The inspired concentration of the potent anesthetic is gradually increased 0.5% every three to four breaths until anaesthesia is induced. Raising the concentration at this rate generally leads to a smooth induction of anaesthesia. Increasing the concentration more rapidly for an agent with a pungent odor, such as halothane, may irritate a patient's airway and cause coughing. Conversely, a slower rate of increase prolongs induction time and makes patients vulnerable to all the problems, such as airway obstruction and vomiting, that could arise during a prolonged excitement period. After anaesthesia is induced, the potent inhalation agent concentration is reduced to a maintenance concentration while the intravenous line is secured.

If vital signs become abnormal at any time during the induction period, the concentration of the potent anesthetic is reduced or it is discontinued and the circuit flushed with 100% oxygen. If the oxygen saturation declines, nitrous oxide is discontinued and 100% oxygen is

administered until the oxygen saturation returns to normal while dealing with the cause of the desaturation. Often there is mild to moderate airway obstruction due to collapse of the hypopharyngeal structures of the development of mild laryngospasm. It is recommended that a precordial stethoscope be placed over the larynx at the sternal notch, thus providing immediate input into the early stages of upper airway events. Generally this airway obstruction is readily relieved by gently obtaining a tight mask fit, slightly closing the pop-off valve so as to generate 5 to 10 cm positive end-exmpiratory pressure, and simply allowing the distending pressure of the bag to stent open the airway. In most cases, there is no need to squeeze the bag.

Isoflurane is a more pungent drug tan halothane; because of its pungency, inducing anaesthesia in unpremedicated patients by slowly increasing the concentration of the drug is more difficult. A isoflurane induction of anaesthesia is associated with a several-fold higher incidence of airway problems and is generally not recommended. If isoflurane must be used, increasing the concentration gradually (0.25 to 0.5% increase in concentration every 10 breaths) seems to reduce the propensity to airway irritation.

The induction, recovery, and safety characteristics of sevoflurane compared to halothane with and without nitrous oxide have been evaluated extensively. There is little doubt that the gaseous induction of anaesthesia is more rapid and that the increase in the inspired sevoflurane concentration can proceed more rapidly than with halothane. Sevoflurane has replaced

halothane in many institutions where cost is not a factor, whereas others limit its used to the induction period, changing to halothane or isoflurane for the maintenance of anaesthesia.

Inhalation Induction

Children acceptability with Inhalation Induction can be improved by having transparent mask or scented transparent mask. The child can allowed to set up during the Induction of anaesthesia or he or she can be allowed to hold his or her mask during Induction of anaesthesia placing a mask directly on the face of the child is often frightening. If the flow rate of the Inspired gas is 10L, This rate exceeds that of the child. Therefore the child will quickly go to sleep without the sensation of being smothered. When the child loses consciousness the mask can be applied tenthly to his or her face.

For over three decades, halothane was the standard Inhalation induction agent in paediatrics, It offered reasonably fast loss of consciousness with minimal airway Irritation. Despite its tendency to slow the heart rate and in the presence of hypercarbia to predispose the child to arrhythmia's it was and is the "gold standard" against which other inhalation agents are compared.

Indicators of Wakefulness

Although complete recovery of mental acuity after an inhalation anesthetic may take 1 to 4 days, much of our concern in the PACU focuses on the more rapid return of the protective reflexes necessary for cardiorespiratory stability. These reflexes include the ability to prevent

airway obstruction resulting from posterior displacement of the tongue, epiglottis, and soft palate or from secretions, the ability to expel tracheobronchial secretions by coughing, baroreceptor reflexes to support perfusion, and chemoreceptor reflexes to support respiration in response to hypercarbia or hypoxemia. The principles governing recovery from inhalation anaesthesia are well summarized by Eger. For inhalation anesthetics, 50% of patients have been shown to respond to a simple command such as "open your eyes" at 20% to 60% of the minimum alveolar concentration (MAC) of the anesthetic; such patients are said to be MAC-awake. Achieving MAC-awake may also be confounded by interactions. a general clinical impression is that adults at MAC-awake levels of anaesthesia (if unimpeded by other factors) are able to maintain and protect their airways. A similar impression in children recovering from anaesthesia is the spontaneous eye opening predicts safe airway maintenance - that is, intact airway reflexes.

Physicochemical Factors Governing Recovery from Inhalation Anaesthesia

When inhalation anaesthesia is discontinued, the rate of decline of alveolar concentration of the gas is a function of alveolar ventilation, anesthetic solubility (blood/gas solubility coefficient, or λ), cardiac output, and the venous-to-alveolar partial pressure difference. Increased ventilation results in a more rapid decline in alveolar anesthetic concentration, which hastens recovery, provided that the arterial carbon dioxide pressure is not so low that it diminishes cerebral blood flow and the removal of anesthetic

agent from the brain. The blood/gas solubility coefficient is an important determinant of recovery time. A MAC-awake state is generally reached within 2 minutes after the discontinuation of nitrous oxide; in contrast, reaching such a state may take several hours after discontinuation of a highly soluble agent such as ether. The λ, in order of decreasing solubility is ether (12), halothane (2.5), isoflurane (1.4), sevoflurane (0.65), nitrous oxide (0.47), and desflurane (0.45). Thus for the potent inhalation agents currently used, the time required for patients to reach a MAC-awake state (when no other sedating medications have been administered) are generally intermediate between that for nitrous oxide and ether.

Maintenance of high anesthetic concentrations results in greater accumulation of the anesthetics in tissues and prolongs recovery time. The higher the λ, the more drug is taken up into body tissues and therefore the longer it takes for the drug to redistribute out of tissue upon discontinuing anaesthesia. The duration of anaesthesia therefore affects recovery, and this effect is more pronounced the more soluble the anesthetic agent. For example, maintenance of halothane anaesthesia at 1.1 MAC for 15 minutes results in recovery to a MAC-awake state in approximately 4 minutes, whereas such recovery may take 15 minutes after 2 hours of anaesthesia at the same MAC.

However, despite an apparent shorter period for awakening with both sevoflurane and desflurane compared with halothane or isoflurane, this does not result in a more rapid discharge from the hospital or recovery room. This may be a result of other factors such as local practice, emergence delirium,

the need to treat pain, administration of other sedating medications, and nursing or parental issues unrelated to anesthetic management.

CAUDAL EPIDURAL ANAESTHESIA

The caudal approach to the epidural space in children was first described in 1933 by Campbell. Today it is the most popular regional block performed in peadiatric patients in the operating room. A single shot caudal block is easy to perform, reliable, and safe with a high degree of success in children less than 7 years of age. It is quick and efficient means of producing perioperative analgesia in children undergoing surgery for genital, lower abdominal and lower limb operations. The block is usually performed after an inhalational or intravenous induction of anaesthesia, but before surgery, to decrease the intraoperative opiod/anesthetic requirements and to provide excellent postoperative analgesia.

Anatomy of the Caudal/Epidural Space in Children

Caudal Space

In the infant the sacrum is a triangular bone formed by the fusion of the five sacral vertebrae. It is not completely ossified at birth and appears relatively wide and shallow. By the age of 8 years these vertebral arches become completely ossified and unite with one another and with the vertebral bodies.

The sacral hiatus, which results from the non-fusion of the fifth sacral vertebral arch, is situated at the lower end of the sacrum, covered by the

sacrococcygeal membrane. It is located between the two bony prominences known as the sacral cornuea. These landmarks are sometimes visible in thin children and are always easily palpable through the skin due to the absence of the large sacral pad of fat that develops at puberty. Maldevelopment of the sacral canal roof occasionally results in significant variations in anatomy of the sacral hiatus and may be the cause of caudal block failures in a small percentage of children. In newborns andinfants, the sacrum is made up of cartilage and soft tissue, making unintended needle entry into bone and rectum possible during caudal block injection. Since the sacrum is situated higher in relation to the illia than in the adult, the sacral hiatus in young children appears higher (cephalad) than expected in adults. The spinal cord and the dura mater end at L3 and S3, respectively, at birth, and at L1 and S2, respectively, in adults. The dural sac extends down to the S4 foramen in children versus S2-S3 in adults and there is no cerebrospinal fluid (CSF) below this sacral level. Anomalies in the position of the dural sac reflection can result in unintended dural puncture.

Epidural Space

The epidural space surrounds the spinal meninges and extends from the Foramen Magnum, where the dura is fused to the base of the skull, to the sacral hiatus. It contains nerve roots, fatty tissue, lymphatics, arteries, veins and connective tissue.The epidural veins are important in the absorption of drugs from epidural space. They are valveless and form a part of the internal vertebral venous plexus that communicates with thoracic and abdominal veins. Therefore, the effective blood volume of the epidural space will

diminish with increased abdominal pressure, necessitating a smaller dose of local anesthetic and a slower rate of injection.

The epidural fat in the newborn and in younger children (< 8 years of age) has a spongy, gelatinous appearance with distinct spaces between the fat lobules. This allows the easy spread of local anesthetics injected into the epidural space and fairly reliable cephalad advancement of a caudal catheter inserted via the sacral hiatus. By contrast, the adult and order child's epidural space has fat lobules that are densely packed and surrounded by fibrous strands, thus preventing drug spread and/or Cephald advancement of the catheter.

Epidural nerve roots arise from the spinal cord enclosed by a short cuff of meninges that contains a pocket of spinal fluid separated from the epidural space by a greatly thinned dura. The diffusion of drugs occurs across the dura into the spinal fluid and into the dorsal nerve roots at this area. Spinal nerve roots serve motor, sensory and autonomic functions. Sensory afferent impulses can be blocked or modified at the level of the roots or the cord.

Physiology of Caudal / Epidural Blockade in the Padiatric Patient

Local anesthetics block nerve impulse transmission by blocking the sodium permeability at specific ion-selective sodium channels and thus prevent the propagation of action potentials. To reach the sodium channel, the local anesthetics must first cross the nerve's outer surface to reach the inner core. Therefore, the thicker and more myelinated motor fibers are more

difficult to block than the thinner less myelinated sensory and sympathetic nerve fibers (A-delta, and C fibers). Since the spread of local anesthetics and opioids is limited by large myelinated nerves, the largest nerve roots C7-8 and S1 are more resistant to blockade via the epidural route. Blockade of sensory nerves reduces peripheral sensation, blocks visceral pain fibers and decreases the neurohumoral response to surgical stimulation within the blocked segment.

The thoraco-lamber symphathetic outflow at spinal levels T1 to L2 exerts control of the central and peripheral vasculature. Blockade of the sympathetic nervous system (sympathctomy) results in hypotension due to arterial and venous dilation. Normally, changes in the venous tone are compensated for by the baroreceptors that reflexly increase the vascular tone peripherally and the heart rate centrally. There is, therefore, an increase in vasoconstrictor activity in the upper extremities to compensate for vasodilation in the lower limbs. High segmental levels of blockade (T2-4), however, prevent not only peripheral vasoconstriction but also the reflex tachycardia.

Hypotension due to sympathetic blockade from epidurally administered local anesthetics is seen commonly in older children and adults and is unusual in the very young presumed to be due to immature sympathetic nervous system in infants.

Clinical Applications

Single-Shot Caudal Technique

Caudal blocks in children are performed in anesthetized or heavily sedated patients once intravenous access has been obtained. The child is usually positioned in the lateral decubitus position. The coccyx is first identified by applying firm pressure between the buttocks with the index finger of the non-dominant hand. It is best to palpate by moving the finger from side to side gently in a cephalad direction. The paired bony protuberances that are first encountered on either side are the two sacral cornuae surrounding the sacral hiatus. The skin over the caudal area is carefully prepared with an antiseptic solution and asepsis is maintained by wearing sterile gloves or by palpating the skin through a sterile alcohol swab (the notouch technique). After identifying the sacral hiatus, the caudal space is entered using a short (1-inch), 20-gauge short bevel needle that has been attached to a labeled syringe containing the appropriate volume and concentration of the local anesthetic solution to be administered. The needle must be placed precisely in the midline and inserted at a 45-60 degree angle to the coronal plane, perpendicular to all other planes and maintaining a rostral direction.

An important landmark to keep in mind during a caudal block is an imaginary line drawn through the two S2 foramina that are immediately below and medial to the posterior superior iliac spines. This is the level at which the dural sac ends and a needle placed above this level risks accidental dural puncture. Care must be taken to position the bevel of the needle facing anteriorly in order to minimize the chance of piercing the anterior sacral wall if the sharp point of the needle is introduced first, which is a very common cause of aspirating blood. As the needle is advanced, a

distinct "pop" is felt on piercing the sacrococcygeal membrane. The needle is immediately lowered to a 10-20 degree angle and simultaneously advanced a few mm cephalad to ensure the entry of the entire bevel surface into the caudal space. If a syringe has to be attached or changed after the needle has entered the caudal space, the use of a slip tip syringe instead of a Luer-lock syringes will decrease the change of unwanted needle movement. After careful aspiration to test for absence of blood or CSF, the appropriate amount of local anesthetic is injected in 1-cc increments. The patients is then turned to the supine position and monitoring of vital signs continued.

Measures to Ensure Effectiveness of a Caudal Block

The three important variables determining the quality and level of caudal blockade are the volume, does and concentration of the injected drug. The rostral spread of caudal analgesia depends on the volume of local anesthetic injected. There are several formulae for determining the volume-to-level relationship for caudal anaesthesia in children. Most are too complex for routine clinical use. According to Armitage, bupivacaine 0.25% in volumes of 0.5 ml/kg. 1 ml/kg and 1.25 ml/kg will provide analgesia to sacral, lower thoracic and mid-thoracic dermatomes respectively. When this formula results in volume of local anesthetic greater than 20ml, he suggests decreasing the bupivacaine concentration to 0.20% in order to prevent the unpleasant sensation of lower limb weakness or heaviness.

Pediatric Caudal Dosage

(0.25% bupivacaine)

Lumbosacral 0.5 ml/kg

Thoraco-lumbar 1.0 ml/kg

Mid-thoracic 1.25 ml/kg

Tests to Confirm Effectiveness of Caudal Blockade

It is difficult to assess the effectiveness of the caudal block by testing for sensory levels in an anesthetized child. The ability to decrease the concentration of potent inhaled anesthetic agent without using opioids is often considered to indicate a successful block. Laxity of the patient's anal sphincter, as evidenced during placement of an acetaminophen suppository after the block had set in, can be used as a test to confirm the effectiveness of the caudal block in children. The presence of a lax anal sphincter at the end of surgery correlates significantly with the reduced administration of opioids intraoperatively and in the PACU. A tight sphincter at the end of surgery may suggest the need to repeat the block before the child awakens, or consider alternative methods of postoperative analgesia. A repeat caudal block is safe if the duration of surgery has been more than 90-120 minutes. The "Swoosh" test a modification of the "Whoosh" test described in adults, has been used in children to confirm successful caudal placement. Instead of injection of air (which produces the "whoosh" sound) the local anesthetic is injected into the caudal space in children to elicit the "Swoosh" sound, which can be heard by placing a stethoscope over the lower lumbar spine.

REVIEW OF LITERATURE

The Laryngeal mask airway has greatly modified airway management since it was first described by Brain in 1983. The laryngeal mask airway is easy to position and provides a secure airway for both spontaneously breathing patients and those whose lungs are mechanically ventilated.

Complications associated with the use of laryngeal mask airway (LMA) have been reported during insertion, maintenance and during recovery.

Gataure PS Latto IP; Rust S et al - Can. J Anaesthesialogy 1995 Dec 42(12) 1113- 6.

Studied the Complication associated with removal of the Laryngeal mask airway (LMA) in deeply aneasthetized state versus awake state in urological patients.

They concluded that it may be safer to remove the laryngeaol mask airway while the patients are deeply anaesthetized in the operating room than they are awake in the recovery room.

Baird MB; Mayor AH; Goodwin A.P.C. et al European Journal of Anaethesiology volume - 16, Number 4, April 1999, 251 - 6.

They compared the incidence of undesirable respiratory events when the Laryngeal mask airway is removed from paediatric patients who are fully awake or deeply anaesthetized .

They found out that oxygen desaturation in children of less than 6 years of age (SaO_2 < 96%) occured most frequently after awake removal (31.3%) compared with deep anaesthesia removal (45%) p = 0.023.

Kitching AJ, Walpole AR, Blog CE et al Br J Anaesthesiology 1997 Mar;78(3) : 337-8 made a comparative study of laryngeal mask airway removal between anaesthetized and awake children. They observed that significant difference exists between the two groups for coughing (p < 0.001) with greater incidence (17 of 33) in awake group compared with those in whom Laryngeal mask airway was removed while anaesthetized (two of 27). There were no difference in the Incidences of laryngospasm, oxygen desaturation (< 95%) and excess salivation between the groups. Removal of laryngeal mask airway during deep anaesthesia reduced coughing in the immediate post operative period.

RI Patel,RS Hannallah, (et al)

Emergence airway complications in children: a comparison of tracheal extubation in awake and deeply anesthetized patients Anesthesia & Analgesia, Vol 73, 266-270. The found oxygen saturation levels were higher in deep extubation than in awake at 1, 2, 3, and 5 min. There were no differences between the two groups in the number of patients requiring supplemental oxygen.

Xiao W, Deng X.

The minimum alveoloar concentration of enflurane for laryngeal mask airway extubation in deeply anesthetized children. Anesth Analg. 2001 Jan; 92(1): 72-5.

They concluded thee may be fewer problems associated with the laryngeal mask airway extubation when patients are deeply anesthetized.

Pappas Ac, Sunhani R, Lurie J, Pawlowsci, J Sawicki K, Corsino A. J.Clinical anaesthesiology 2001 Nov 13(7) 498-503 compared the influence of anaesthetic depth and choice of volatile anaesthesia drug on the incidence and severity of airway hyper reactivity associated with Laryngeal mask airway removal in children. They found out severe airway hyperreactivity leading to a critical event ie partial or complete laryngospasm with oxygen saturation $SPO_2 < 85\%$ was only encountered in patients maintained with isoflurane and laryngeal mask airway removed when child awakened. Adverse airway events $SPO_2 < 90\%$, Vomiting and bronchospasm were also significantly higher in above said Group.

Pander, K, Bluck stockd, Steward Dj et al 1991 Anaesthesiology Tracheal extubation in children; Halothoane Vs isoflurane, anaesthetized Vs awake. They concluded awake tracheal extubation following either agent was associated with significantly more episode of oxygen desaturtion than the tracheal extubation while anaesthetized.

Gregory Ca, Eger EI, Munsun et al The relationship between age and halothane requirement in man 1969 30 : 488 - 491 They showed that

data for humans derived from a comprehensive Literate Survey were consistent for age > 1 yr with log MAC decreasing with increasing age at the same rate for all inhaled anasthetics approximately equivalent to 6% change per decade of age.

Mason dg, Bingham rm et al Laryngeal mask airway in children Anaestheiology 1990 Sep 45(9) : 760-3. They proved that clear airway was achieved in more than 95% case and concluded that size 2 laryngeal mask airway can be successfully used in weight range of 6 - 30 kg.

Johnston DF, Wrigley SK, Robb PJ, Jones hf Anaesthesia 1990 Nov 45 (11) 924-7. The study showed Laryngeal mask airway produces satisfactory airway in all children.

Lopez - gill M, Brimacombe J, Alvarez et al Safety and efficacy of laryngeal mask airway. They documented LMA mask airway provides safe and effective form of airway management in infants and children.

Mamaya B. Paediatric anaesthesia 2002 June 12(5) Airway management in Spontaneously breathing anaesthetized children comparing the Laryngeal mask airway with the cuffed oro pharyngeal airway. They proved more positional manoeuvers required to achieve a satisfactory airway with Copa.

Reignier J, Ben Ameur M, Ecoffey C. Spontaneous Vertilation with halothane in children. A comparative study between Endotracheal tubve and laryngel mask airway.

Anaesthesiology 1995; 83 : 674 - 678. Their study showed that pardoxical inspiratory movement is less when breathing though an laryngeal mask airway than through an endotracheal tube.

Jehan M, Kamal abdel - halim, Mauda shouley azer and Ghada ahmed.

Journal of Egyptean National Cancer Institute Vol. 14 No.4 Dec 319 - 323 2002.

Conclusion of this study was that Sevoflurane and halothane were similar in the smoothness of induction and recovery and the quality of anaesthesia overall in children.

Fujii Y ; Saitoh Y; Tanaka it ; Toyooka H ; Cardiovascular response to tracheal extubation of Laryngeal mask airway removal in children Canadian Journal of anaesthesia 1998 45 - 2.

Showed that Laryngeal mask airway removal elicited less hemodynamic changes than tracheal extubation in Paediatric patients.

Reddy Sv, Win N, Singapore medical journal 1990 : 31

The found out that post OP complication in awake LMA removal includeds clenching of teeth 10%, Vomitus, 4% excessive salivation 6%..

O'Neill B, templeton, caramito, Schreinar MS Anaesthesia Analgesia 1994; 78; 659 - 662.

Has documented coughing on emergence and showed 2% Lidocaine topical solution decreased the incidence of Coughing on emergence.

Springer Dk, Jahr Js, American Journal of Anaesthesiology 1995:22:65-69 has confirmed the safety and efficacy of Laryngeal mask airway.

Joshi Gp, Inagkic Y, White Pf Anaesthesia Analgesia 1998 Oct 87(4) has found Laryngeal mask airway as an alternative to traditional intubation in ambulatory anaesthesia.

MATERIALS AND METHODS

The study was conducted in forty boys aged between 1-12 years in whom general anaesthesia combined with regional block was appropriate. This is a randomised, single blind study design.

Local ethics committee approval was obtained. Informed written consent obtained from parents.

Exclusion Crtieria

1. Anaesthesia within 3 months.
2. Current or chronic upper airway disease.
3. Asthma.
4. Congenital heart disease.

Study patients were divided in to

- **Awake group (A)**
- **Deeply Anaesthetized Group (D)**

Intravenous line with balanced salt solution was established in all patients.

Premedication

- Im atropine 10 mics / kg
- Im Midazolam 0.05 mg / kg

  30 - 45 minutes before surgery

Monitoring

- Precordial Stethoscope
- Pulse oximetry
- Electrocarduogram
- NIBP monitor
- A rolled up gauze swab served as a bite block untel the LMA was removed.

Induction

Patients were preoxygenated for 3 minutes. Oxygen and nitrous oxide were administered in the ratio of 2:3. Halothane started with 0.5% and increased up to 4% in increments of 0.5%. The duration between increments is approximately 30 seconds.

Conditions for LMA Insertion was assessed by eyelash reflex and jaw relaxation. Iv fentanyl 1mics /kg was given two minutes before insertion of Laryngeal mask airway. LMA was then Introduced using the standard technique. This technique involves a completly deflated LMA held like a pen guided in to the pharynx with the Index finger of the operator at the junction of the tube and the bowl with the operator at the head of the patient and the LMA aperture facing caudally with the head extended and the neck flexed by using the hand under the occiput, under direct vision the top of the cuff is pressed upwards against the hard palate. The LMA is advanced in to the hypopharynx till a resistance is felt. The cuff is then Inflated just enough air to seal, using the air volume for that age. Position of the LMA checked. Bilateral air entry found to equal.

By holding the tube in position, patient was turned in to left lateral position. Caudal epidural given by palpating the sacral hiatus. A 22 gauge needle is used to pierce sacro coccygeal membran. Aspiration for blood and CSF was done. After negative aspiration 0.5 ml of 0.25% Bupivacaine given

Maintenance

Anaesthesia was maintained with appropriate concentration of oxygen, Nitrous oxide and halothane. oxygen saturation was maintained at or above 95% during operation.

Removal of LMA

Five minutes before the anticipated end of surgery, anaesthesia was deepened by giving twice the minimum alveolar concentration (MAC) of halothane (adjusted for age)

The child was then allocated randomly by tossing a coin to have the LMA removed and replaced with a Guedel airway (deep group) or left in-situ (awake group).

Each child was than turned onto the left side and transferred to the recovery room with supplemental oxygen 4 litre min^{-1} by face mask.

Diclofenac 12.5 mg (or 25 mg if the child weighed more then 20 cm) was inserted per rectum on arrival in the recovery room.

Monitoring the patient

A pulseoximeter probe was applied to a finger of the uppermost hand and haemoglobin oxygen saturation and heart rate were desplayed.

Continuous close observation were made of coughing and straining, laryngospasm, Bronchospasm, oxygen de-saturation (<95%) vomiting and

salivation for 60 minutes from the end of anaesthesia or the child was awake, whichever was earlier.

Observations and management were recorded. The LMA or guedel airway was removed in the recovery room when the child regains full airway reflexes.

OBSERVATION AND RESULTS

We studied 40 children undergoing urological surgeries in the age group of 1 to 12 years.

Out of the 40 children 18 children had their LMA removed in deep plane of anaesthesia (Deep Group) and 22 when they were awake (Awake group). The number of patients with airway complication in the study group on whole is 22 (55%). The airway parameters that were included in the study are

1. coughing and straining
2. oxygen desaturation ($SPO_2 < 95\%$)

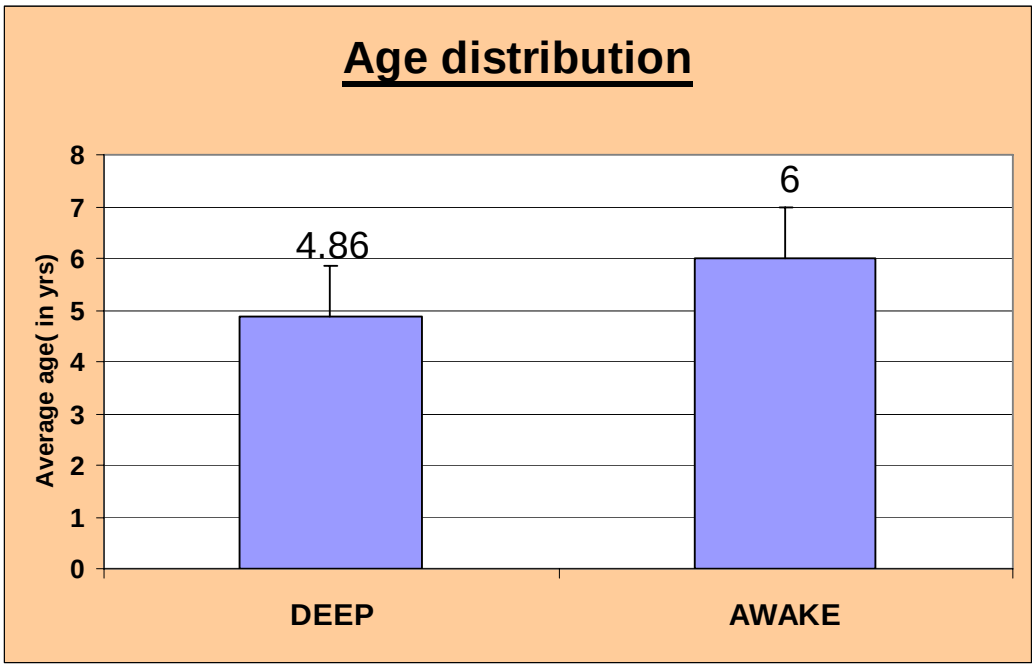
3. laryngospasm

4. Bronchospasm

5. Excessive salivation and vomiting

TABLE 1

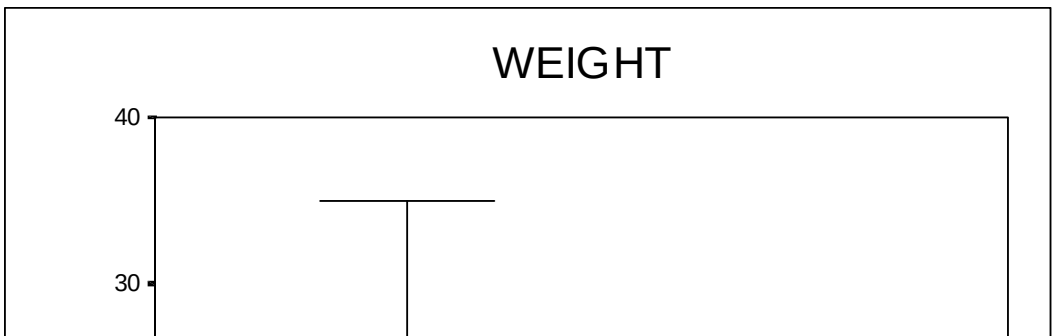
AGE					
	Group	N	Mean	Std. Deviation	Student t Test
Age/Sex	Awake	22	6.00	4.183	t= 1.03
	Deep	18	4.86	2.331	P = 0.30



The mean age of patients in deep group (De) is 4.86 years and that of awake group (Aw) is 6.00 yrs.

TABLE 2

WEIGHT					
	Group	N	Mean	Std. Deviation	Student t Test
WT (kg)	Awake	22	17.45	7.249	t = 1.62 P = 0.10
	Deep	18	14.72	5.345	

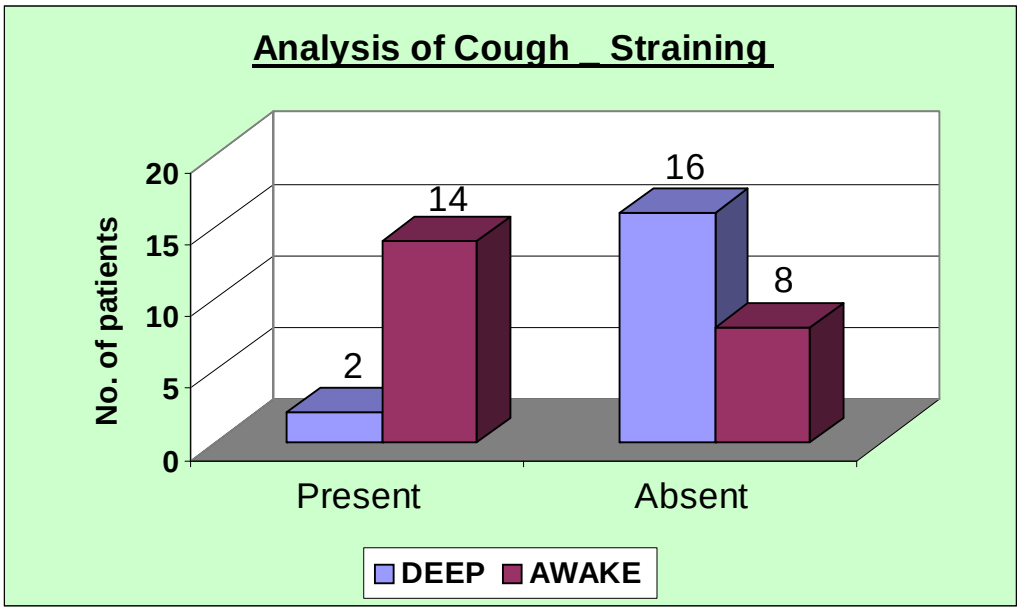


The mean weight of patients in deep group (D) is 14.72kg and awake (A) group is 17.45kg.

TABLE 3

		Coughing and straining		Total
		Absent	Present	
Group	Awake	8	14	22
	Deep	16	2	18
Total		24	16	40

χ^2 Yates = 9.3 P = 0.001 OR (95% Cl) = 14 (2-11)

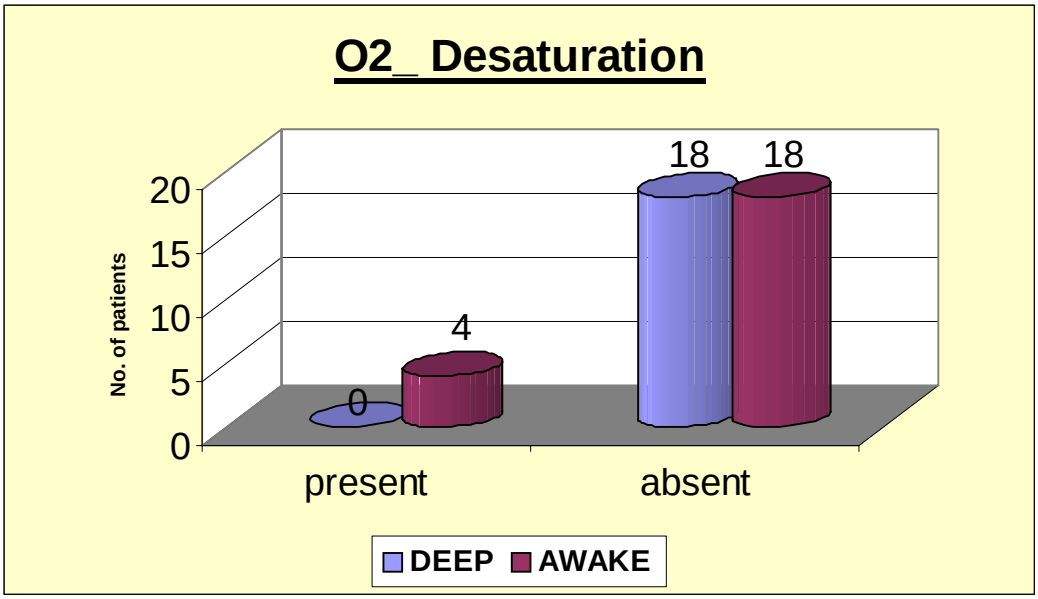


In awake group 14 patients had coughing and straining, only 2 patients in deep group had coughing and straining. Awake group patients has 14 times more chance of coughing and straining then the deep group As P = 0.001 this is statistically significant.

TABLE 4

		Oxygen de-saturation		Total
		Absent	Present	
Group	Awake	18	4	22
	Deep	18	0	18
Total		36	4	40

χ^2 Yates = 3.63 P = 0.07 NOT SIGNIFICANT



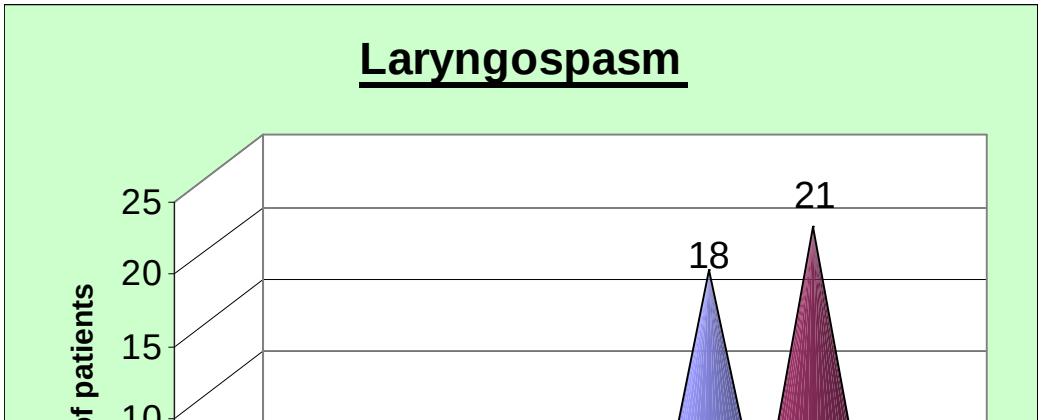
Four patients had oxygen destruction in the awake group as compared with none in deep group.

As P = 0.07 it is statistically Insignificant.

TABLE 5

		Laryngospasm		Total
		Absent	Present	
Group	Awake	21	1	22
	Deep	18	0	18
Total		39	2	40

χ^2 Yates = 1.72 P = 0.19 NS

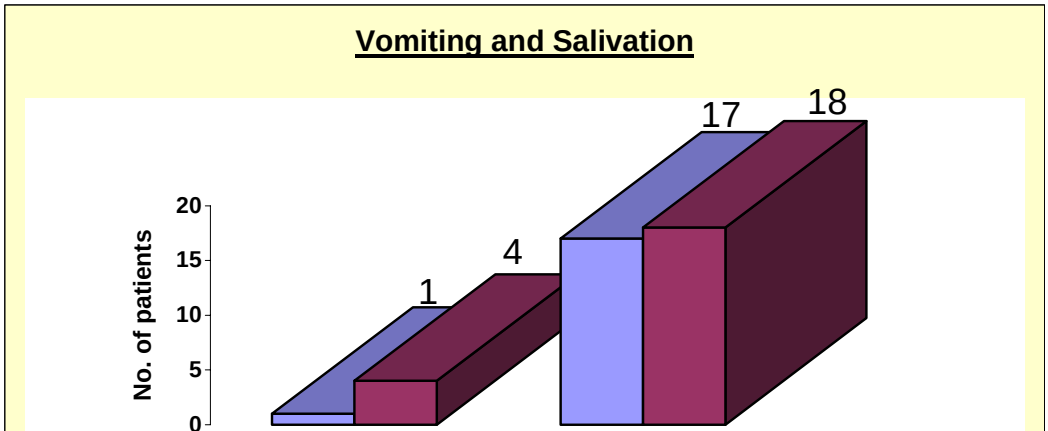


Only one patient had experienced mild laryngospasm which was treated by mask ventilation with 100 % 02. This patient belongs to awake group. Coughing preceded this event and LMA was removed to prevent excessive coughing. Patient had normal breathing after three minutes As P = 0.19 that is statistically Insignificant.

TABLE 6

		Vomiting and Salivation		Total
		Absent	Present	
Group	Awake	18	4	22
	Deep	17	1	18
Total		36	4	40

χ^2 Yates = 0.72 P = 0.06 NOT SIGNIFICANT



Four Patients in awake group and one patient in deep group had excessive salivation. No one had vomiting. As P = 0.06, it is statistically Insignificant. Excessive salivation is defined as enough salivation to coat a finger.

Bronchospasm was not encountered during the whole study. In deep group three patients had airway complication. Out of the three patients two had coughing and straining, which occurred 5 mins after LMA removal. Both had only one episode of coughing and straining. Neither patient in this group who had coughed had O_2 saturation values less than 95%. Only one patient had significant salivation.

In awake groups, airway complication occurred in 14 patients. Fourteen patients had coughing and straining. Out of which ten had more than one episode these then children started coughing and straining before they were awake ie (response to command and purposeful voluntary movement and spontaneous Eye opening) and it necessitated the removal of LMA in the recovery room.

Remaining Patient LMA were removed after they were awake. The LMA was removed by anesthesiologist who is monitoring the patient. Heart rare increased during LMA removal in all patients.

Statistical Analysis

Qualitative variables were given in frequencies with their percentage.

Quantitative variables were given in mean and standard deviation.

Qualitative data were analyzed using Recuson Chi-squared Test and odds ratio were given with 95% confidence interval.

Quantitative data were analyzed using student t-test

P - value Less 0.05 was taken as statistically significant

DISCUSSION

Laryngeal mask airway has become a integral part of Anaesthesiologist armentarium many studies have been done for insertion of LMA relatively few studies have come for "LMA removal". The aim of the study is to find out optimal time for LMA removal in paediatric patients.

The incidence of airway complications on removal of LMA in this study was 11.11% in the deep group and 63.63% in the awake group ($p < 0.01$).

The patients for this study were children presenting for short urological procedures. Most of the surgeries were of less than one hour duration. Shorter duration surgeries require minimal duration of halothane exposure so recovery can be quicker and smoother. .

Halothane was selected as induction agent so that a standard anaesthetic regimen were followed to ensure that the groups were as comparable as possible, intravenous induction agents were not used because it effect on airway irritability have individual variation.

IV fentanyl is given 2 minutes before LMA insertion to suppress airway reflexes in all patients .After LMA insertion caudal extradural block given to provide analgesia to the operative site. This was done to eliminate airway irritability due to pain from the operative site and therefore airway complications were likely to be secondary to the local effect of LMA.

The entire anaesthetic technique was designed to make the time of removal of LMA the only variable.

Patients were exposed to two MAC of halothane approximately 5 minutes before the end of surgery to ensure that both group receive the same amount of volatile anaesthetic.

Laffon et al reported a two-fold increased incidence of complications after removal of LMA in awake compared with deeply anaesthetized paediatric patients. This study shows that it is not always possible to leave in LMA in site until the patient is fully awake ie patients having spontaneous eye opening, purposeful voluntary movements and return of full airway reflexes.

When the child regains the swallowing reflex as noted by movement of deglutition they are unable to tolerate the laryngeal mask airway cuff in the pharynx. This irritability leads to coughing and straining. Return of

swallowing also drives the pharyngeal secretions to the glottis to induce coughing as the cuff seal around the laryngeal inlet is not 100% protective.

Parry m, glaisyer h.r, p.bailey p.m. et al., proved that swallowing does not equate with either full return of airway reflexes or an awake state. The return of the swallowing in the anaesthetized patient with the LMA in situ is associated with an inadequate or light depth of anaesthesia. So we were forced to remove the LMA at too early a stage in recovery process and because of this it associated with increased complications such as coughing and straining. Cuff was deflated before LMA removal in all cases.

Mason and Bingham et al reported that removal of LMA is the only action required for airway complication like coughing and straining that occurs in paediatric patients with LMA in the recovery room.

In this study out of 22 awake paediatric patients, 10 patients the LMA was removed when swallowing reflexes returned. This swallowing reflex does not equate with full return of airway reflexes thereby provoking increased airway complications.

Failure to prevent Coughing and straining in recovery period can be a problem after urological procedures, the induced increase in venous pressure can lead to oozing from the suture site and even haematoma formation,which could impair the viability of sutured tissue .

Therefore for paediatric patients we advocate removal of the LMA in deep plane to remove the stimulating effect on airway

It is been suggested that LMA can be removed "deep" or at an "awake" plane but coughing is most likely in awake plane. As we have inferred coughing and straining can be detrimental to specific urological and plastic procedures.

SUMMARY

In paediatric patients when the LMA was removed while they were awake, 63.66% had coughing and straining and this is statistically significant (p=0.001). Even though coughing and straining in the awake group patients is not significantly associated with other respiratory events like oxygen desaturation, bronchospasm or laryngospasm but coughing and straining induced increase in venous pressure and heamotoma formation may be undesirable in specific urological and plastic procedures. To avoid this complication paediatric patients LMA can be safely removed in deep plane without any adverse respiratory events. Hence Paediatric patients LMA can be removed safely in deep plane for some specific procedures.

CONCLUSION

Normally LMA is removed once the patient regains airway reflex fully but in specific urological and plastic surgical procedures, even slightest coughing and straining on the LMA can lead to detrimental effort on the issue flap which could impair the out come of surgery. To avoid this complication the LMA can be removed in deep plane. From this study it is concluded that the paediatric patients LMA can be removed safely in deep plane without any adverse respiratory events.

PROFORMA

REMOVAL OF LMA IN CHILDREN

DEEPLY ANAESTHETIZED COMPARED WITH AWAKE

Name : Age / Sex : WT(kg) :

Diagnosis : IP No. : Date:

Planned Procedure :

Pre OP Evaluation

BP mm / Hg

PR / min

ASA Classification :

MPC Classification :

EXCLUSION CRITERIA

- Anaesthesia within 3 months
- Current or Chronic airway disease
- Asthma
- Congenital heart disease

Study Group (A) - **Awake Removal**

Study Group (D) - **Deep Removal**

Premedication	-	IM midazolam 0.05 mg/kg 30 mins before surgery IM Atropine 10 mics / kg 30 mins before surgery
Monitoring		ECG, NIBP, Pulse Oximetry

INDUCTION

Halothane, Nitrous Oxide and Oxygen, Fentanyl 1 mics/kg (Before Insertion)

LMA Insertion : Difficult / Easy

Rescue Drug	:	Fentanyl 1 mics / kg
Supplemental RA	:	Caudal Epidural 0.25% Bupivacaine 0.5 ml /kg.
Maintenance	:	O2 (50%), Nitrous Oxide 50%, Halothane
Deepening of Anaesthesia	:	5 Mins before anticipated closure, twice the MAC of halothane adjusted for age Diclofenac 12.5 mg PR given before shifting to recovery room. (25 mg Diclofenac if > 20 kg)

INCIDENCE OF AIRWAY EVENTS

Sl. No.	**Airway Events**	**Study Group**	**No. of Episodes**	**Timing of Episodes**
1	Coughing & Straining			
2	Oxygen desaturation			
3	Bronchospasm			
4	Laryngospasm			
5	Vomiting / Salivation			

BP / PR CHART

LMA Removal	5 Mins Before	Immed After	5 Mins	10 Mins	15 Mins	20 Mins	25 Mins
BP							
PR							

MASTER CHART

DEEPLY ANAESTHETIZED

Sl. No.	Name	Age / Sex	WT (Kg)	C & S	BH	O2D	BS	LS	V/s
1	Kowshik	2.5 Mch	15	Nil	Nil	Nil	Nil	Nil	Nil
2	Mohan Kumar	3.5 Mch	15	Nil	Nil	Nil	Nil	Nil	Nil
3	Vignesh	1.5 Mch	12	Nil	Nil	Nil	Nil	Nil	Nil
4	Rohit	2 Mch	11	Nil	Nil	Nil	Nil	Nil	Nil
5	Vimal	3 Mch	12	Nil	Nil	Nil	Nil	Nil	Nil
6	Kamalakannan	3 Mch	10	Nil	Nil	Nil	Nil	Nil	Nil
7	Richardson	4 Mch	16	Nil	Nil	Nil	Nil	Nil	Nil
8	Guruprasad	2 Mch	12	Nil	Nil	Nil	Nil	Nil	Nil
9	Vijaya Shanthi	6 Mch	16	Nil	Nil	Nil	Nil	Nil	Nil
10	Kumaran	7 Mch	23	1	Nil	Nil	Nil	Nil	Nil
11	Dhanush Kumar	1.5 Mch	10	Nil	Nil	Nil	Nil	Nil	Nil
12	Dheena	5 Mch	15	Nil	Nil	Nil	Nil	Nil	1

13	Boopalan	6 Mch	17	1	Nil	Nil	Nil	Nil	Nil
14	Md.Asif	2 Mch	10	Nil	Nil	Nil	Nil	Nil	Nil
15	Pasupathy	6 Mch	18	Nil	Nil	Nil	Nil	Nil	Nil
16	Ahmed Jalal	10 Mch	31	Nil	Nil	Nil	Nil	Nil	Nil
17	Tamilmani	1.75 Mch	12	Nil	Nil	Nil	Nil	Nil	Nil
18	Yogesh	2.5 Mch	10	Nil	Nil	Nil	Nil	Nil	Nil

C & S Cough & Straining

ml mild laryngospasm

O2D Oxygen desaturation

BS Bronchospasm

LS Laryngospasm

V/S Vomiting / Salivation

1 one episode

s significant salivation

AWAKE

Sl. No.	Name	Age / Sex	WT (Kg)	C&S	O2D	BS	LS	V/s
1	Prasanna	12/Mch	22	3	Nil	Nil	Nil	Nil
2	Dhanush	2/Mch	10	2	1	Nil	Nil	Nil
3	Yuvaraj	5/Mch	18	1	Nil	Nil	Nil	Nil
4	Forizon	11/Mch	28	2	Nil	Nil	Nil	Nil
5	Guhan	1/Mch	9	Nil	Nil	Nil	Nil	1
6	Prakash	7/Mch	22	1	Nil	Nil	Nil	Nil
7	Sanjay Kumar	12/Mch	26	4	1	Nil	ml	1
8	Muthuraj	2.5/Mch	13	3	1	Nil	Nil	Nil
9	Ajay	12/Mch	27	Nil	Nil	Nil	Nil	Nil
10	Sivarama Krishnan	5/Mch	15	2	Nil	Nil	Nil	Nil
11	Sanjay Krishna	2.5/Mch	12	Nil	Nil	Nil	Nil	Nil
12	Mohan	11/Mch	28	2	Nil	Nil	Nil	1
13	Sadag Ali	12/Mch	35	1	Nil	Nil	Nil	Nil
14	Arasamuthu	11/Mch	25	Nil	Nil	Nil	Nil	Nil

15	Vijaya Baskar	1.25/Mch	13	1	Nil	Nil	Nil	Nil
16	Kalaiarasan	5/Mch	14	Nil	Nil	Nil	Nil	Nil
17	Samy	3/Mch	13	Nil	Nil	Nil	Nil	Nil
18	Yesunath	1.5/Mch	11	2	Nil	Nil	Nil	Nil
19	Sabarisan	2/Mch	11	Nil	Nil	Nil	Nil	Nil
20	Naveen	3/Mch	15	2	Nil	Nil	Nil	1
21	Pasupathy	6/Mch	19	Nil	Nil	Nil	Nil	Nil
22	Shyam	4/Mch	20	2	1	Nil	Nil	Nil

C & S	Cough & Straining	1	one episode
ml	mild laryngospasm		
O2D	Oxygen desaturation	s	significant salivation
BS	Bronchospasm		
LS	Laryngospasm		
V/S	Vomiting / Salivation		

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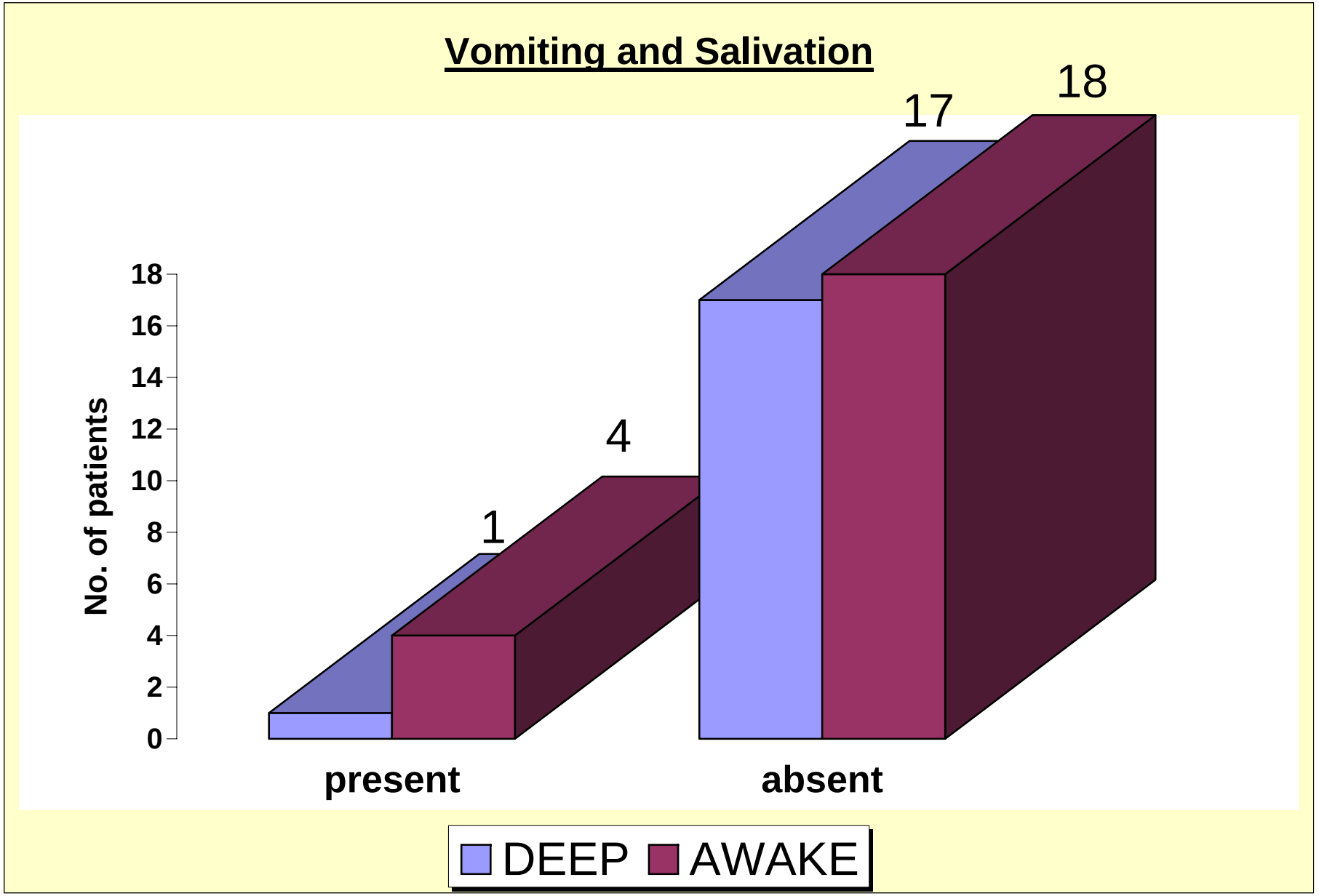
Vomiting and Salivation
18
17
4
1
No. of patients
18
16
14
12
10
8
6
4
2
0
present
absent
DEEP
AWAKE